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| DB = PGPB, USPT, USOC, EPAB, JPAB, DWPI, TDBD; PLUR = YES; OP = OR' | | | | |
| | L45 | L43 and PARP-1 | 5 | |
| | L44 | L43 and nucleolin | 13 | |
| | L43 | L42 and (apoptotic)adj(bodies) | 1352 | |
| | L42 | L(detection)adj(assays) | 5896538 | |
| | L41 | (apoptosis)adj(detection)adj(assays) | 34 | |
| | L40 | L38 and (nucleolin)adj(antibod?) | 0 | |
| | L39 | L38 and (PARP-1)adj(antibod?) | 0 | |
| | L38 | L37 and blood | 28 | |
| | L37 | (apoptosis)adj(detection)adj(assays) | 34 | |
| | L36 | (PARP)same(apoptotic)adj(bod?) | 4 | |
| | L35 | (nucleolin)same(apoptotic)adj(bodies) | . 2 | |
| | L34 | (nucleolin)same(serum)same(plasma)same(blood) | 10 | |
| | DB=EPA | AB; PLUR=YES; OP=OR | | |
| | L33 | WO-9603655-A1.did. | 1 | |
| | DB=PG | $PB, USPT, USOC, EPAB, JPAB, DWPI, TDBD; \ PLUR = YESCOMBON $ | S; OP = OR | |
| | L32 | L29 and anti-PARP | 0 | |
| | L31 | L29 and anti-nucleolin | . 0 | |
| | L30 | L29 and nucleolin | 0 | |
| | L29 | (circulating)adj(apoptotic)adj(bodies) | 12 | |
| | L28 | L27 and nucleolin | 6 | |
| | ·L27 | (530/387.1).ccls. | 2729 | |
| | L26 | L22 and anti-nucleolin | . 1 | |
| | L25 | L22 and anti-PARP | 7 | |
| | L24 | L23 and apoptosis | . 16 | |
| | L23 | L22 and nucleolin | 39 | |
| | L22 | (435/7.1).ccls. | 12323 | |
| | L21 | (mi)adj(yingchang) | 3 | |
| | L20 | (yingchang)adj(mi) | 0 | |
| | L19 | (bates)adj(paula)adj(j) | 9 | |
| | L18 | (detection)same(apoptosis)same(anti-nucleolin) | 1 | |
| | L17 | L16 and (uncleaved) | 9 | |
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| L16 | (detection)same(apoptosis)and anti-PARP | 83 |
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| L15 | (detection)same(apoptosis)same(PARP-1) | 2 |
| L14 | (detection)same(apoptosis)same(nucleolin) | . 3 |
| L13 | L11 and anti-PARP | 38 |
| L12 | L11 and (anti-nucleolin) | 1 |
| L11 | L10 and apoptosis | 1356 |
| L10 | (apoptotic)adj(bodies) | 1464 |
| L9 | (nucleolin)same(antibod?)same(apoptosis) | . 5 |
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| . L8 | WO-200061597-A1.did. | 0 |
| DB=DW | VPI; PLUR=YES; OP=OR | |
| L7 | 200061597 | 2 |
| DB=US | PT; PLUR=YES; OP=OR | |
| L6 | 6339075.pn. | 1 |
| L5 | 6325785.pn. | 1 |
| L4 | 6291643.pn. | 1 |
| L3 | 6048703.pn. | 1 |
| L2 | 5932475.pn. | 1 |
| L1 | 5925334.pn. | 1 |

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=> s apoptotic bodies

L1 7817 APOPTOTIC BODIES

=> s l1 and blood

L2 1025 L1 AND BLOOD

=> s 12 and plasma

L3 120 L2 AND PLASMA

=> s 13 and serum

L4 34 L3 AND SERUM

=> s 14 and detection

L5 12 L4 AND DETECTION

=> s 15 and nucleolin

L6 1 L5 AND NUCLEOLIN

=> d l6 cbib abs

ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN Document No. 140:90312 A method for the detection of 2004:20982 apoptosis via determination of nucleolin and/or PARP-1 in the sample. Bates, Paula J.; Mi, Yingchang (University of Louisville Research Foundation, Inc., USA). PCT Int. Appl. WO 2004003554 A1 20040108, 66 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US20167 20030626. PRIORITY: US 2002-392143P 20020626.

AB Methods for the detection of apoptosis by measuring apoptotic bodies shed by apoptotic cells are provided, as are kits to carry out such methods. The method comprises preparing a sample from which cells have been removed and determining at least one of nucleolin and PARP-1 in the sample. The detection of either (or both) compds. comprises the detection of a

nucleolin (PARP-1)-binding mol.-nucleolin (PARP-1) complex. The binding mols. are anti-nucleolin (anti-PARP-1) antibodies and for nucleolin also guanosine-rich oligonucleotides. The sample can be blood, serum, plasma, tissue, tissue culture medium, or sputum. The method can be used to determine excessive apoptosis via preparing a blood sample from a subject suspected of having a disease selected from the group consisting of AIDS, neurodegenerative disease, ischemic injury, autoimmune disease, tumor, cancer, viral infection, acute inflammation, and sepsis.

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L7 8 DUP REMOVE L5 (4 DUPLICATES REMOVED)

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L7 ANSWER 1 OF 8 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

2006496393 EMBASE Circulating nucleic acids in plasma/serum and tumor progression: Are apoptotic bodies involved?

An experimental study in a rat cancer model. Samos J.; Garcia-Olmo D.C.; Picazo M.G.; Rubio-Vitaller A.; Garcia-Olmo D.. Prof. D. Garcia-Olmo, Servicio de Cirugia General-C, Hospital Universitario La Paz, Paseo Castellana 261, 28046 Madrid, Spain. damian.garcia@uam.es. Annals of the New York Academy of Sciences Vol. 1075, pp. 165-173 2006. Editor: Swaminathan R.

Refs: 31.

ISSN: 0077-8923. E-ISSN: 1749-6632. ISBN: 157331627X. CODEN: ANYAA Pub. Country: United States. Language: English. Summary Language: English. Entered STN: 20061027. Last Updated on STN: 20061027

AΒ The "genometastasis hypothesis" proposes that cell-free tumor nucleic acids might be able to transform host stem cells, and that this might be a pathway for the development of metastases. This theory is supported by previous experimental findings and is consistent with observations of other authors. It has been suggested that tumor DNA might be horizontally transferred by the uptake of apoptotic bodies and initiate the genetic changes that are necessary for tumor formation. addition, apoptotic bodies have been proposed as possible vehicles that protect the nucleic acids circulating in the plasma from enzymatic degradation. In the present study, we analyzed the presence of apoptotic bodies in serum and its relationship with tumor progression in a heterotopic model of colon cancer in the rat. We injected DHD/K12-PROb cancer cells subcutaneously into BD-IX rats and divided the animals into three groups according to the time between the injection of tumor cells and euthanasia. A control group of healthy animals was included (n = 6). After euthanasia, macroscopic metastases were assessed and samples of blood were collected. To detect apoptotic bodies in the sera, each sample was mixed with FITC-conjugated annexin V antibody in combination with propidium iodide and then analyzed by flow cytometry. Detection of apoptotic bodies was only significantly increased in the sera of a few tumor-bearing animals in late stages of tumor development. Thus, such particles appear not to be the vehicle of the cell-free tumor nucleic acids that are detected at early stages of cancer. .COPYRGT. 2006 New York Academy of Sciences.

L7 ANSWER 2 OF 8 MEDLINE on STN DUPLICATE 1
2006670327. PubMed ID: 17108207. Circulating nucleic acids in
plasma/serum and tumor progression: are
apoptotic bodies involved? An experimental study in a
rat cancer model. Samos Julia; Garcia-Olmo Dolores C; Picazo Maria G;
Rubio-Vitaller Antonio; Garcia-Olmo Damian. (Experimental Research Unit,
General University Hospital of Albacete, Albacete, Spain.) Annals of the

New York Academy of Sciences, (2006 Sep) Vol. 1075, pp. 165-73. Journal code: 7506858. ISSN: 0077-8923. Pub. country: United States. Language: English.

- The "genometastasis hypothesis" proposes that cell-free tumor nucleic AB · acids might be able to transform host stem cells, and that this might be a pathway for the development of metastases. This theory is supported by previous experimental findings and is consistent with observations of other authors. It has been suggested that tumor DNA might be horizontally transferred by the uptake of apoptotic bodies and initiate the genetic changes that are necessary for tumor formation. addition, apoptotic bodies have been proposed as possible vehicles that protect the nucleic acids circulating in the plasma from enzymatic degradation. In the present study, we analyzed the presence of apoptotic bodies in serum and its relationship with tumor progression in a heterotopic model of colon cancer in the rat. We injected DHD/K12-PROb cancer cells subcutaneously into BD-IX rats and divided the animals into three groups according to the time between the injection of tumor cells and euthanasia. A control group of healthy animals was included (n = 6). After euthanasia, macroscopic metastases were assessed and samples of blood were collected. To detect apoptotic bodies in the sera, each sample was mixed with FITC-conjugated annexin V antibody in combination with propidium iodide and then analyzed by flow cytometry. Detection of apoptotic bodies was only significantly increased in the sera of a few tumor-bearing animals in late stages of tumor development. Thus, such particles appear not to be the vehicle of the cell-free tumor nucleic acids that are detected at early stages of cancer.
- L7 ANSWER 3 OF 8 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN 2006:279900 Document No.: PREV200600286904. Method enabling use of extracellular RNA extracted from plasma or serum to detect, monitor or evaluate cancer. Kopreski, Michael S. [Inventor]. Portage, MI USA. ASSIGNEE: Oncomedx, Inc.. Patent Info.: US 06939671 20050906. Official Gazette of the United States Patent and Trademark Office Patents, (SEP 6 2005) CODEN: OGUPE7. ISSN: 0098-1133. Language: English.
- This invention relates to the use of tumor-derived or associated AB extracellular ribonucleic acid (RNA) found circulating in the plasma or serum fraction of blood for the detection, monitoring, or evaluation of cancer or premalignant conditions. Extracellular RNA may circulate as non-bound RNA, protein-bound RNA, lipid-RNA complexes, lipoprotein (proteolipid)-RNA complexes, protein-RNA complexes including within or in association with ribonucleoprotein complexes, nucleosomes, or within apoptotic bodies. Any intracellular RNA found in plasma or serum can additionally be detected by this invention. Specifically, this invention enables the extraction of circulating RNA from plasma or serum and utilizes nucleic acid amplification assays for the identification, detection, inference, monitoring, or evaluation of any neoplasm, benign, premalignant, or malignant, in humans or other animals, which might be associated with that RNA. Further, this invention allows the qualitative or quantitative detection of tumor-derived or associated extracellular RNA circulating in the plasma or serum of humans or animals with or without any prior knowledge of the presence of cancer or premalignant tissue.
- L7 ANSWER 4 OF 8 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN 2006:243150 Document No.: PREV200600252319. Method enabling use of extracellular RNA extracted from plasma or serum to detect, monitor or evaluate cancer. Kopreski, Michael S. [Inventor]. Long Valley, NJ USA. ASSIGNEE: OncoMEDx, Inc.. Patent Info.: US 06916634 20050712. Official Gazette of the United States Patent and Trademark Office Patents, (JUL 12 2005)

- CODEN: OGUPE7. ISSN: 0098-1133. Language: English. This invention relates to the use of tumor-derived or associated AΒ extracellular ribonucleic acid (RNA) found circulating in the plasma or serum fraction of blood for the detection, monitoring, or evaluation of cancer or premalignant conditions. Extracellular RNA may circulate as non-bound RNA, protein-bound RNA, lipid-RNA complexes, lipoprotein (proteolipid)-RNA complexes, protein-RNA complexes including within or in association with ribonucleoprotein complexes, nucleosomes, or within apoptotic bodies. Any intracellular RNA found in plasma or serum can additionally be detected by this invention. Specifically, this invention enables the extraction of circulating RNA from plasma or serum and utilizes nucleic acid amplification assays for the identification, detection, inference, monitoring, or evaluation of any neoplasm, benign, premalignant, or malignant, in humans or other animals, which might be associated with that RNA. Further, this invention allows the qualitative or quantitative detection of tumor-derived or associated extracellular RNA circulating in the plasma or serum of humans or animals with or without any prior knowledge of the presence of cancer or premalignant tissue.
- ANSWER 5 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN Document No. 140:90312 A method for the detection of 2004:20982 apoptosis via determination of nucleolin and/or PARP-1 in the sample. Bates, Paula J.; Mi, Yingchang (University of Louisville Research Foundation, Inc., USA). PCT Int. Appl. WO 2004003554 A1 20040108, 66 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US20167 20030626. PRIORITY: US 2002-392143P 20020626.
- AΒ Methods for the detection of apoptosis by measuring apoptotic bodies shed by apoptotic cells are provided, as are kits to carry out such methods. The method comprises preparing a sample from which cells have been removed and determining at least one of nucleolin and PARP-1 in the sample. The detection of either (or both) compds. comprises the detection of a nucleolin (PARP-1)-binding mol.-nucleolin (PARP-1) complex. The binding mols. are anti-nucleolin (anti-PARP-1) antibodies and for nucleolin also guanosine-rich oligonucleotides. The sample can be blood, serum, plasma, tissue, tissue culture medium, or sputum. The method can be used to determine excessive apoptosis via preparing a blood sample from a subject suspected of having a disease selected from the group consisting of AIDS, neurodegenerative disease, ischemic injury, autoimmune disease, tumor, cancer, viral infection, acute inflammation, and sepsis.
- L7 ANSWER 6 OF 8 MEDLINE on STN DUPLICATE 2
 2004090891. PubMed ID: 14718398. Characterization of amplifiable,
 circulating RNA in plasma and its potential as a tool for cancer
 diagnostics. El-Hefnawy Talal; Raja Siva; Kelly Lori; Bigbee William L;
 Kirkwood John M; Luketich James D; Godfrey Tony E. (Division of Thoracic
 Surgery, School of Medicine, Hillman Cancer Center, University of
 Pittsburgh, Pittsburgh, PA 15213, USA.) Clinical chemistry, (2004 Mar)
 Vol. 50, No. 3, pp. 564-73. Electronic Publication: 2004-01-12. Journal
 code: 9421549. ISSN: 0009-9147. Pub. country: United States. Language:
 English.
- AB BACKGROUND: Several recent reports have described the detection of circulating, cancer-related RNA molecules in serum or

plasma from cancer patients, but little is known about the biology of this extracellular RNA. We aimed to determine how RNA is protected against degradation in serum, to optimize RNA isolation from large volumes of serum, and to test our optimized assays for serum-based cancer detection. METHODS: We used quantitative reverse transcription-PCR (QRT-PCR) analysis to investigate the isolation and biology of extracellular plasma RNA. We then examined the presence of amplifiable RNA transcripts in plasma and serum from controls and from patients with esophageal cancer and malignant melanoma. RESULTS: We found that extracellular RNA in plasma is highly degraded and can be isolated most efficiently by guanidinium-phenol extraction followed by precipitation. Extracellular RNA is stable in serum for up to 3 h but is destroyed immediately by addition of detergents. Extracellular RNA can be captured on 0.2 microm filters, allowing concentration of RNA from several milliliters of plasma. When we concentrated RNA from up to 4 mL of serum, detection of cancer-related transcripts in serum from cancer patients and controls was infrequent and inconsistent. CONCLUSIONS: Extracellular RNA is most likely protected within protein or lipid vesicles, possibly apoptotic bodies, which can be disrupted by detergents. Despite optimizing many aspects of plasma RNA detection, we were unable to reproducibly detect cancer-related transcripts. Our data suggest that measurement of circulating RNA may not be a good approach to early cancer diagnosis.

- L7 ANSWER 7 OF 8 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN 2002:113843 Document No.: PREV200200113843. Method enabling use of extracellular RNA extracted from plasma or serum to detect, monitor or evaluate cancer. Kopreski, Michael S. [Inventor, Reprint author]. Long Valley, NJ, USA. ASSIGNEE: OncoMEDx, Inc., Long Valley, NJ, USA. Patent Info.: US 6329179 20011211. Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 11, 2001) Vol. 1253, No. 2. http://www.uspto.gov/web/menu/patdata.html. e-file. CODEN: OGUPE7. ISSN: 0098-1133. Language: English.
- AB This invention relates to the use of tumor-derived or associated extracellular ribonucleic acid (RNA) found circulating in the plasma or serum fraction of blood for the detection, monitoring, or evaluation of cancer or premalignant conditions. Extracellular RNA may circulate as non-bound RNA, protein-bound RNA, lipid-RNA complexes, lipoprotein (proteolipid) -- RNA complexes, protein-RNA complexes including within or in association with ribonucleoprotein complexes, nucleosomes, or within apoptotic bodies. Any intracellular RNA found in plasma or serum can additionally be detected by this invention. Specifically, this invention enables the extraction of circulating RNA from plasma or serum and utilizes nucleic acid amplification assays for the identification, detection, inference, monitoring, or evaluation of any neoplasm, benign, premalignant, or malignant, in humans or other animals, which might be associated with that RNA. Further, this invention allows the qualitative or quantitative detection of tumor-derived or associated extracellular RNA circulating in the plasma or serum of humans or animals with or without any prior knowledge of the presence of cancer or premalignant tissue.
- L7 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

 1997:650277 Document No. 127:315563 Extraction, amplification, and detection of extracellular tumor-derived RNA from plasma or serum to detect, monitor or evaluate cancer. Kopreski, Michael S. (Kopreski, Michael S., USA). PCT Int. Appl. WO 9735589 A1 19971002, 54 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG,

US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1997-US3479 19970314. PRIORITY: US 1996-14730 19960326. AΒ This invention relates to the use of tumor-derived or associated extracellular RNA found circulating in the plasma or serum fraction of blood for the detection, monitoring, or evaluation of cancer or premalignant conditions. Extracellular RNA may circulate as non-bound RNA, protein-bound RNA, lipid-RNA complexes, lipoprotein (proteolipid)-RNA complexes, protein-RNA complexes including within or in association with ribonucleoprotein complexes, nucleosomes, or within apoptotic bodies. Any intracellular RNA found in plasma or serum can addnl. be detected by this invention. Specifically, this invention enables the extraction of circulating RNA from plasma or serum and utilizes nucleic acid amplification assays for the identification, detection, inference, monitoring, or evaluation of any neoplasm, benign, premalignant, or malignant, in humans or other animals, which might be associated with that RNA. Further, this invention allows the qual. or quant. detection of tumor-derived or associated extracellular RNA circulating in the plasma or serum of humans or animals with or without any prior knowledge of the presence of cancer or premalignant tissue. In a typical example, keratin 19 mRNA is extracted from blood serum using the silica extraction method, followed by RT-PCR amplification with ELISA detection; pos. testing for extracellular keratin 19 mRNA suggests an impending cancer recurrence in a woman who had been treated for breast cancer two years ago.

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PROCESSING COMPLETED FOR L8
L9 2 DUP REMOVE L8 (0 DUPLICATES REMOVED)

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ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN Document No. 140:90312 A method for the detection of apoptosis 2004:20982 via determination of nucleolin and/or PARP-1 in the sample. Bates, Paula J.; Mi, Yingchang (University of Louisville Research Foundation, Inc., USA). PCT Int. Appl. WO 2004003554 Al 20040108, 66 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US20167 20030626. PRIORITY: US 2002-392143P 20020626.

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neurodegenerative disease, ischemic injury, autoimmune disease, tumor, cancer, viral infection, acute inflammation, and sepsis.

ANSWER 2 OF 2 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN 2003:201624 Document No.: PREV200300201624. Apoptosis in leukemia cells is accompanied by alterations in the levels and localization of nucleolin. Mi, Yingchang; Thomas, Shelia D.; Xu, Xiaohua; Casson, Lavona K.; Miller, Donald M.; Bates, Paula J. [Reprint Author]. 570 S. Preston St., 204B Baxter Bldg., Louisville, KY, 40202, USA. paula.bates@louisville.edu. Journal of Biological Chemistry, (March 7 2003) Vol. 278, No. 10, pp. 8572-8579. print. CODEN: JBCHA3. ISSN: 0021-9258. Language: English. Molecular defects in apoptotic pathways are thought to often contribute to AΒ the abnormal expansion of malignant cells and their resistance to chemotherapy. Therefore, a comprehensive knowledge of the mechanisms controlling induction of apoptosis and subsequent cellular disintegration could result in improved methods for prognosis and treatment of cancer. In this study, we have examined apoptosis-induced alterations in two proteins, nucleolin and poly(ADP-ribose) polymerase-1 (PARP-1), in U937 leukemia cells. Nucleolin is expressed at high levels in malignant cells, and it is a multifunctional and mobile protein that can shuttle among the nucleolus, nucleoplasm, cytoplasm, and plasma membrane. Here, we report our findings that UV irradiation or camptothecin treatment of U937 cells induced apoptosis and caused a significant change in the levels and localization of nucleolin within the nucleus. Additionally, nucleolin levels were dramatically decreased in extracts containing the cytoplasm and plasma membrane. These alterations could be abrogated by pre-incubation with an inhibitor of PARP-1 (3-aminobenzamide), and our data support a potential role for nucleolin in removing cleaved PARP-1 from dying cells. Furthermore, both nucleolin and cleaved PARP-1 were detected in the culture medium of cells undergoing apoptosis, associated with particles of a size consistent with apoptotic bodies. These results indicate that nucleolin plays an important role in apoptosis, and could be a useful marker for assessing apoptosis or

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L11 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN 2004:20982 Document No. 140:90312 A method for the detection of apoptosis via determination of nucleolin and/or PARP-1 in the sample. Bates, Paula J.; Mi, Yingchang (University of Louisville Research Foundation, Inc., USA). PCT Int. Appl. WO 2004003554 A1 20040108, 66 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US20167 20030626. PRIORITY: US 2002-392143P 20020626.

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L13 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
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L21 ANSWER 1 OF 3 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

2006:609547 The Genuine Article (R) Number: 055SJ. Proliferating effects of the flavonoids daidzein and quercetin on cultured chicken primordial germ cells through antioxidant action. Tang X Y; Zhang C Q (Reprint); Zeng W D; Mi Y L; Liu H Y. Zhejiang Univ, Dept Vet Med, Coll Anim Sci, 268 Kaixuan Rd, Hangzhou 310029, Zhejiang, Peoples R China (Reprint); Zhejiang Univ, Dept Vet Med, Coll Anim Sci, Hangzhou 310029, Zhejiang, Peoples R China. cqzhang@zju.edu.cn. CELL BIOLOGY INTERNATIONAL (MAY 2006) Vol. 30, No. 5, pp. 445-451. ISSN: 1065-6995. Publisher: ACADEMIC PRESS LTD ELSEVIER SCIENCE LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND. Language: English.

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

Primordial germ cells (PGCs) are undifferentiated pluripotent stem AB cells, whose proliferation is influenced by many internal and external factors. In the present study, a PGC-somatic cell co-culture model was established to evaluate effects of the flavonoids daidzein (DAI) and quercetin (QUE) on proliferation of PGCs from embryonic chickens. PGCs were isolated from the germinal ridge of 3.5-4 day embryos and cultured in 5% fetal calf serum (FCS)-supplemented Medium 199. PGC subculture was carried out on chicken embryonic fibroblast feeder (CEF) or follicular granulosa cell feeder (GCF) layers. The subcultured PGCs were challenged with flavonoids alone or in combination with a reactive oxygen substance (ROS)-producing system on CEF for 48 h. The results showed a better supporting effect of CEF than GCF. Flavonoids (1 mu g/ ml) significantly promoted PGC proliferation, which could be markedly inhibited by ROS. The oxidative damage by ROS was further manifest by decreased superoxide dismutase activity and glutathione levels. In addition, activation of protein kinase A (PKA) by forskolin significantly stimulated PGC proliferation, but PKA inhibitor H89 inhibited the proliferating effects induced by DAI and QUE. These results indicated that cultured PGCs respond to exogenous agents on proliferation and that antioxidant flavonoids could restore the intracellular antioxidant system and promote PGC proliferation via their antioxidant action involving the PKA signaling pathway. (c) 2006 International Federation for Cell Biology. Published by Elsevier Ltd. All rights reserved.

L21 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN 2004:20982 Document No. 140:90312 A method for the detection of

apoptosis via determination of nucleolin and/or PARP-1 in the sample. Bates, Paula J.; Mi, Yingchang (University of Louisville Research Foundation, Inc., USA). PCT Int. Appl. WO 2004003554 A1 20040108, 66 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US20167 20030626. PRIORITY: US 2002-392143P 20020626.

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MEDLINE on STN L21 ANSWER 3 OF 3 DUPLICATE 1 2004456047. PubMed ID: 15364206. Effects of follicle-stimulating hormone and androgen on proliferation of cultured testicular germ cells of embryonic chickens. Mi Yuling; Zhang Caiqiao; Xie Meina; Zeng Weidong. (Department of Veterinary Medicine, College of Animal Sciences, Zhejiang University, Hangzhou 310029, China.) General and comparative endocrinology, (2004 Sep 15) Vol. 138, No. 3, pp. 237-46. Journal code: 0370735. ISSN: 0016-6480. Pub. country: United States. Language: English. A germ-Sertoli cell coculture model was established to study effects of AΒ follicle-stimulating hormone (FSH) and testosterone (T) on testicular germ cell proliferation of the embryonic chickens. Germ and somatic cells were dispersed from 18-day-old embryonic testes and cultured in 96-well plates. Germ cells were characterized by expression of stem cell factor receptor c-kit. Germ cell proliferation was assessed by an increase in cell number and expression of proliferating cell nuclear antigen (PCNA). Results showed that the germ and Sertoli cells kept alive in serum-free McCoy's 5A medium supplemented with insulin, transferrin, and selenite (ITS medium). Germ cells adhered to the free surface of Sertoli cells that spread the filopodia and formed a monolayer in ITS medium. In the serum-containing medium, Sertoli cells displayed an increment with a flat squamous form and only a few very large germ cell masses were found in the free surface of Sertoli cells. Many germ cells showed apoptosis in the McCoy's 5A medium without ITS or serum. Only germ cells showed positive staining for c-kit in the coculture. Ovine FSH (0.25-1.0 IU/ml) significantly increased the number of germ cells, and PCNA-labeling index (P < 0.05). FSH also induced stronger c-kit expression compared with the control. In the FSH-treated groups, germ cells were manifested distinct knob-like form. Similar stimulating effect was found in the germ cell number by T treatments (10(-7)-10(-6)M). Furthermore, FSH (0.5 IU/ml) combined with T significantly promoted higher testicular germ cell proliferation (P < 0.05) compared with either FSH or T alone, which indicated that interaction of FSH and T might be additive. The above results showed that the serum-free germ-Sertoli cell coculture model allowed evaluating hormonal regulation of testicular germ cell proliferation. FSH and T promoted testicular germ cell proliferation probably through indirect effects on Sertoli cells.

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L23 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN Document No. 140:90312 A method for the detection of 2004:20982 apoptosis via determination of nucleolin and/or PARP-1 in the sample. Bates, Paula J.; Mi, Yingchang (University of Louisville Research Foundation, Inc., USA). PCT Int. Appl. WO 2004003554 Al 20040108, 66 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC; LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US20167 20030626. PRIORITY: US 2002-392143P 20020626.

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2003099862. PubMed ID: 12506112. Apoptosis in leukemia cells is
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Mi Yingchang; Thomas Shelia D; Xu Xiaohua; Casson Lavona K; Miller
Donald M; Bates Paula J. (Molecular Targets Group, James Graham
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Kentucky 40202, USA.) The Journal of biological chemistry, (2003 Mar 7)
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| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
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| FULL ESTIMATED COST | 117.45 | 117.66 |
| DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) | SINCE FILE ENTRY | TOTAL SESSION |
| CA SUBSCRIBER PRICE | -7.80 | -7.80 |

STN INTERNATIONAL LOGOFF AT 12:57:08 ON 22 JAN 2007